REMARKS

Claims 1 and 2 have been amended in response to the Examiner's comments to emphasize that both the composition and method of treatment of the invention reduce the lesion size resulting from cerebral ischemia. Claim 3 has been cancelled without prejudice. No 'new matter' has been added.

The rejection of claims 1 and 2 under 35 USC 102 as anticipated by Pratt et al. is respectfully traversed.

As even more clearly set forth in the amended claims, the present invention is directed to the use of enoxaparin to reduce the lesion size (and thereby minimize neuronal damage) in the treatment of cerebral ischemia. As discussed in previous prosecution, this is a complex pathological condition, generally caused by reperfusion, and linked to a total or partial loss of vascularization of a brain area. The deprivation of oxygen and the resulting inflammation and biochemichal disorders can lead to cellular death and then to cerebral infarction. Cerebral ischemia is the consequence of a local arterial occlusion or embolism.

By contrast, Pratt et al. (Haemostasis, 1998, 28: 78-85) (hereinafter, 'PRATT') investigated the effect of enoxaparin on edema following a photothrombic injury in the rat. The dosages disclosed by PRATT are irrelevant because they are not set forth in the context of treating lesions caused by cerebral ischemia. While the Examiner has asserted that PRATT inherently discloses the present invention, he has failed to establish a prima facie case of inherency. Quite to the contrary, PRATT is devoid of any disclosure relating to cerebral ischemia. Indeed, the PRATT disclosure and the present invention do not share the same aim at all. Thus, even if one defines the skilled person as an expert in the neurodegenerative diseases art, PRATT does not relate to the resolution of the same problem as the present invention, nor does it contain any mention of the treatment of cerebral ischemia. Further, the focal photothrombotic model used by PRATT is not suitable for provoking a neurological deficit in the rat. Indeed, PRATT does not mention reperfusion, much less the cerebral ischemia that can result therefrom, in its publication.

Accordingly, the cited PRATT reference neither inherently nor explicitly describes (nor suggests) the presently claimed use of enoxaparin in the treatment of cerebral ischemia.

The rejection of Claims 2 and 3 as anticipated by Physician's Desk reference (49th edition, 1995) (hereinafter 'PDR') is similarly traversed, particularly in light of the foregoing amendment of claim 2 and deletion of claim 3. Thus, the PDR reference for Lovenox teaches only one dosage form and one use for Lovenox – treatment or prevention of deep vein thrombosis.

In view of the foregoing, favorable reconsideration and prompt Notice of Allowance are earnestly solicited.

A Notice of Appeal is being filed concurrently herewith.

Respectfully submitted,

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